MATTERS ARISING

Epidemiology of whiplash

Space restrictions prohibit a comprehensive refutation of the uneven treatment of the whiplash literature presented by Ferrari and Russell.[1] They fiercely interrogate research that does not support their view, yet uncritically embrace literature favouring their preconceptions. Central to their argument is the assertion that there are different rates of chronic whiplash in different countries, and that "chronic injury related damage cannot account for the wide differences".

A valid comparison between the prevalence of any condition in two places would require that it is measured in the same way. Balla's study comparing Singapore and Australia was little more than anecdotal from interviews of selected Singaporean doctors compared with the data from Australia.[2] Such data may be fatally corrupted by recall, case selection, sampling, and expectation bias.

Caution should be observed in comparing insurance claim rates between countries. There is no international consistency in notification of accidents or insurance or compensation procedures. Conclusions drawn from such comparisons[3] are unsustainable and subject to the ecological fallacy. The frailty of using insurance claims as a surrogate for the incidence of injury does not seem to have been considered by Ferrari and Russell. A claim is a behaviour arising from a combination of motivation, enabling circumstances, perceived benefits, costs, social norms, peer and family pressure, and fear of current or future pain and disability—all factors extraneous to the injury itself. The Victorian experience in Australia is particularly pertinent. Fewer claims for whiplash were noted after the introduction of legislation creating bureaucratic barriers, disincentives, and upfront costs for potential claimants. Some then concluded that whiplash is a behaviour and not an injury.[4] A more sober view is that if it is harder to make a claim, fewer people will make one. To extrapolate beyond this is unjustifiable: the apparent change in incidence may simply be due to reporting bias.

The Lithuanian study has been used to argue that chronic symptoms after whiplash do not occur in communities lacking a compensation system.[5] However, only 31 patients developed any neck pain as a result of the accident, with none reporting chronic pain. The 95% confidence limits of this estimate range up to 10%. Therefore, the data are consistent with a rate of chronicity of up to 10%. The German and later Lithuanian studies, on which Ferrari and Russell rely, also lack the power to detect a significant chronicity rate.

Magnetic resonance imaging (MRI) is insensitive to abnormalities of the soft tissue components of the cervical zygapophysial joints.[6] Consequently, studies of patients with whiplash who have normal MRIs cannot exclude important injury. Furthermore, both ultrasound[7] and bone scan studies have shown potentially painful pathology.[8]

In considering our studies of chronic zygapophysial joint pain after whiplash, Ferrari and Russell argue that our patients were unrepresentative. However, most of our patients developed pain within 72 hours of the accident and were passengers or drivers of motor vehicles.[9] They were intentionally representative and typical of patients with chronic whiplash. Radanov's work is criticised on the basis that they "selectively gathered 117 patients through advertisement". This would imply that patients answered advertisements if they had whiplash, producing a biased sample. However, the advertisement was in a medical journal, seeking doctors to enrol participants, producing a representative sample. Concurrently, Ferrari and Russell have used these studies in a previous article, apparently accepting the methodology then.[10] These flaws alone raise grounds for concern that the opinions of Ferrari and Russell are not a responsible appraisal of the literature and will raise alarm and reinforce prejudice against genuinely afflicted patients.

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Through their leader, Ferrari and Russell[1] venture to raise alarm about whiplash, repeating the same arguments that they have already raised in two previous editorials and a letter to the editor. But their alarm is overstated and misplaced.

Acute whiplash is not a problem. Even the studies of Radanov *et al* show that only some 5% of patients have severe symptoms at 12 months.[2] Meanwhile, the study of Borchgrevink *et al* sets a benchmark.[3] Most patients can be adequately treated simply by advising them to act as usual. If there is any psychosocial problem with acute whiplash, it is on the part of doctors and therapists who overmedicalise this problem.

However, even so, some 10–20% of patients remained symptomatic at six months.[3] Two questions arise: why are these patients symptomatic, and what should be done about them? Our own approach has been to investigate these patients for a possible source of pain. Under stringent, double blind, controlled conditions we have found that we can pinpoint a source of pain in the zygapophysial joints in some 50% of these patients.[4] Moreover, by surgical treatment we can relieve their pain[5] and their psychological distress[6] and return them to normal life.

These patients may not be typical of acute patients, but they are quite typical of chronic patients. Ferrari and Russell[1] contend that zygapophysial joint pain must be rare. Indeed, it is, for it accounts for only half of 5–10% of the original population; but it accounts for 50% of the chronic population. Elsewhere, Ferrari and Russell[1] deny that persisting symptoms can be attributed to the original whiplash, but this is a legal matter, not a medical one. There are no medical tests by which to falsify an imputation. Ferrari and Russell invoke the studies of Schraeder *et al*[7] to prove that chronic whiplash does not occur. However, they cannot argue from the general to the specific. Indeed, even Schraeder *et al* themselves point out that their results cannot be used to refute an individual claim that their chronic pain resulted from the whiplash.[7]

Ferrari and Russell[1] argue that there is no persisting lesion, and that psychological and social factors totally explain the chronic complaints of these patients. In doing so they criticise the work of Radanov, by claiming that it is "fraught with at least 15 significant methodological flaws". They do not enunciate these flaws but instead cite four references, thereby relying on sophistry to seduce their readers. If these references are consulted, the last three offer no criticism of Radanov. Only the first, a letter, offers criticism, but cleverly Ferrari and Russell[1] do not inform the reader of Radanov's rebuttal of these criticisms.[8]

Yet even if we accept that psychosocial factors are important in these patients, Ferrari and Russell[1] do not provide an answer as to what to do about them. Speciously, they cite van Akkerveeken and Vendrig,[9] but do not explain to readers that this was not a peer reviewed publication, that it was only a preliminary study, that it was not controlled, and that the authors themselves were accordingly guarded about overstating their results. No other literature is provided to vindicate cognitive intervention.

Finally, if Ferrari and Russell are so convinced that experimental studies of whiplash are so innocuous, perhaps they might organise some volunteers to undergo a series of $\Delta V30$ kph and $\Delta V60$ kph collisions, which are what many of our patients underwent.

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Making selective use of the literature and incorrect quoting of previous research, the January 1999 "leader"[1] intends to support the view of the whiplash syndrome as malingering. This reply cannot be exhaustive but will address the following:

The Ballas paper lacked a definition of the whiplash syndrome and did not describe the assessment of 300 selected cases seen in a single practice.[2] Moreover, selection bias applies for the (according to author "anecdotal") control group too.[2] Furthermore, in 20 patients in Singapore with acute whiplash, the injury severity or risk of developing long term symptoms was not specified.[2] Methodological flaws of the Ballas publication[2] are reflected by the fact that this study was not considered relevant by the Quebec Task Force[3] and neither were a number of other references in the "leader".[1] To interpret late whiplash syndrome based on articles such as these is in contradiction to a claim of methodological soundness.[1]

The non-existence of whiplash in the United Kingdom while it has been described for more than 30 years USA[1] is discussed in Miller's 1961 *BMJ* article, which reports 200 cases examined between 1955 and 1957.[4] This is well within the time frame of the 1953 *JAMA* whiplash paper.[5] Miller reported an inverse relation between accident neurosis and the severity of injury and emphasised that the occurrence of "psychoneurosis in patients who were never unconscious was 42%".[4] Reporting on patients who were never unconscious in a concussion series reflects the problems of definition. What was described as whiplash in North America at that time was probably described as concussion in Europe; the problems in defining concussion have been discussed previously.[6] These differences in terminology may be explained by the mechanism of concussion and whiplash, which is the acceleration-deceleration of the head.[7] In addition, symptoms of concussion and whiplash are almost identical.[7] Accordingly, an individual who sustained acceleration-deceleration of the head without loss of consciousness probably has whiplash.

Previously, neck pain in the general population has been reported to vary between 14% in Norway[8] and 33% in Lithuania.[9] These variations were interpreted as "due to sociocultural factors or differences in questioning".[9] It is remarkable that there might be "differences in questioning" as the same researchers participated in both studies.[8 9] However, in large epidemiological studies neck pain is either unreported[11] or the figures are considerably lower[12] than in the Lithuanian studies.[8 9] Accordingly, the method of assessment in the Lithuanian studies[8 9] or reporting of the data might have been biased.

The influence of psychosocial factors, which are secondary to the initial consequences of whiplash (that is, pain), on the further development or increase in symptoms has never been questioned.[13] The "significant methodological flaws or sources of bias" of the Swiss study quoted in the "leader"[1] represent an unwillingness of Ferrari and Russell to analyse in detail results from previous research while continuing to promote their own perspective.[13] In addition, the "leader"[1] emphasised that methodologically improved studies showed "that symptom reporting ... is best predicted by non-accident related stressors". The study quoted in the leader used a biased selection of 39 patients,[13] which was three times fewer than in the Swiss study.

The "leader"[1] emphasised that the Swiss study[14] "selectively gathered 117 patients by advertisement". The truth is that "to obtain a non-selected sample the authors announced the study in the *Swiss Medical Weekly Journal* and repeatedly distributed letters to primary care doctors".[14] Behind this false reporting is probably the hope that the scientific community will eventually become tired of commenting, which eventually may help them to introduce the malingering hypothesis for whiplash injury.

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Authors' reply to Drs Barnsley and Bogduk

We thank Drs Barnsley and Bogduk for their comments. Dr Barnsley reiterates a dualistic (mind-body) approach that we have been trying to blur and indeed do away with for various reasons, most notably that dualistic approaches have been largely unhelpful to date.[1] We do not accept that chronic pain is all in the mind, nor all in the body. We also believe, to use her term, that these patients are "genuinely affected". Dr Barnsley's comment that a "more sober view" suggests a reduction in insurance claims does not reflect a reduction in symptom prevalence requires proof, and is not in accord with admittedly anecdotal reports from Australian rheumatologists, nor with the evidence from Lithuania[2] (she does not quote the subsequent prospective study), Germany,[3 4] and Greece.[5] Dr Barnsley is also well aware of the impressive study presented at the World Whiplash Congress in Vancouver which suggests that changing the claim scheme has dramatic effects on recovery rates, as indicated by various patient centred outcomes.[6]

Both Drs Barnsley and Bogduk have missed the key message in the epidemiological literature—the rapid recovery rate seen in some countries is not being duplicated in others. The studies in Lithuania, Greece, and Germany cannot rule out the possibility of a small number of subjects having chronic pain and disability, but they do show that recovery (as measured by absence of symptoms and return to normal activities, as well as other patient centred outcomes) occurs in 90–95% of subjects in six weeks or less. It is this fact that compels us to question the basis for chronic pain in say, Canada. We find that whiplash in Canada (and reportedly in many other countries) is a massive health and economic burden, with more than 50% of accident victims reporting chronic pain six months after the accident.[6 7] The patients of Dr Bogduk's study represent merely the tip of a large iceberg. Thus new paradigms are necessary to understand why some subjects recover within six weeks or fewer and others do not. As no one has suggested that Lithuanians, Greeks, and Germans have a different anatomy, we need to look for an explanation for this difference in recovery rates.

It is certainly possible that a small proportion of subjects could have chronic structural damage in countries like Lithuania, as Dr Bogduk suggests, and that current studies with background prevalences of neck pain in the control population of up to 10% are not large enough to distinguish an additional 2–3%. Yet, this additional 2–3% of patients are not the group of patients we are describing. It is the 50% of patients with chronic pain at six months[6 7] that we are concerned with, and the cervical zygapophysial studies are not relevant for this larger group. Indeed, we were not aware that the subjects of Dr Bogduk's studies had undergone such high velocity impacts (a ΔV of 30–60 kph) as Dr Bogduk indicates. This fact makes it even less likely that their study group is typical of most patients with chronic whiplash, who instead undergo much lower velocity collisions. Clearly, and for good reasons, Dr Bogduk's study patient spectrum is very different from the group we are concerned with. Our disagreement is not substantially with the few per cent that he may see with facet joint problems, but rather with the rest of the iceberg of chronic pain.

The purpose of our model is to develop discussion on research questions and develop bona fide research efforts to understand what explains different recovery rates, so we can use that understanding in changing both the approach of the therapeutic community and society in response to acute whiplash. Understanding the behaviour that promotes chronic pain is the first, best step to changing it. We agree with Bogduk, once again, that overtreatment and medicalisation are likely to be part of the problem. Yet, until it is thoroughly demonstrated to, and understood by, both the therapeutic community and society at

large, that this is part of the problem, this practice is unlikely to change.

By setting forth this model we can now investigate it. We are making efforts to do this, and we hope that quality researchers such as Drs Barnsley and Bogduk will engage in such efforts as well.

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Authors' reply to Dr Radanov

Dr Radanov's expressed concerns and cry for *auto-da-fé** are based on his perception that our biopsychosocial model is one of malingering as an explanation for the late whiplash syndrome. As we have explicitly stated, in both our current article and in a previous review on this topic, we reject a model based on malingering and we consider this to be a rare or uncommon presentation.[1] Dr Radanov's concerns are therefore misplaced. That Dr Radanov is unable to appreciate how our biopsychosocial model presents alternatives to the otherwise unhelpful, unidimensional, and dichotomous approaches taken by himself and others is a problem for him, but one which we cannot ameliorate in the space available. We thus refer him to a more comprehensive resource.[2]

Once again, we reject the view that the chronic pain of whiplash is due to an enigmatic and inexplicable chronic injury, and we simultaneously reject the view that the best explanation (or even a common explanation) for the late whiplash syndrome is malingering or psychological models that place the pain "all in one's head". The biopsychosocial model includes physical sources for pain, and incorporates psychosocial factors to explain both the severity and attribution of the pain, as well as further behaviour enacted upon this substrate of otherwise benign physical sources of pain. Thus we maintain that the most helpful focus of discussion and research should be on identifying how the various elements of the biopsychosocial model explain the variance in epidemiology of the late whiplash syndrome, and why, even within a given culture some accident victims recover quickly and others do not. Dr Radanov's views may be coloured by the relatively benign nature of the problem he sees in Switzerland. Even with an advertising campaign to recruit subjects, the Swiss outcomes were very much better than those currently being described in North America. We maintain that the Swiss effort at understanding these issues has been a start, but is a mere footstep in a much longer journey of inquisition.

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*Sentence of the Inquisition—burning of the heretic.

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Rheumatoid arthritis, poverty and smoking

Maiden *et al* raise a number of important and interesting points in their paper "Does social disadvantage contribute to the excess mortality in rheumatoid arthritis patients?"[1]

They have observed that mortality in rheumatoid arthritis (RA) correlated with social grouping on the west coast of Scotland. Patients with RA of the lowest socioeconomic classes have an increased mortality when compared with patients of a higher socioeconomic class. Moreover, RA was more prevalent in patients with RA of lower socioeconomic class. We propose that these two important observations can both be explained by cigarette smoking.

The authors commented that cigarette smoking was more prevalent in the patients with RA of lower socioeconomic class in their study. In Britain there is a marked difference in smoking prevalence between social classes. In the 1996 census 41% of lower social class men (social class 4) were current smokers, with only 12% of men in the highest social class (social class 1) currently smoking.[2] Cigarette smoking kills 120 000 people a year in Britain.[3] Most of these deaths are as a result of cardiovascular disease, respiratory disease, and lung cancer. Maiden *et al*[1] observed that 65% of the deaths in their study occurred as a result of these diseases. Current data show that continued cigarette smoking throughout adult life doubles age-specific mortality rates, nearly trebling them in late middle age.[4] Cigarette smoking is associated with an increased risk of RA in both men[5] and women[6]. The increased mortality seen in patients with RA of low socioeconomic status could be explained in part by cigarette smoking, and that cigarette smoking itself might have contributed to the excess RA seen in the most socially deprived.

Since the poorest in our society appear to have an increased risk of RA, studies designed to identify risk factors for RA may best be focused on those with the highest risk. Cigarette smoking may be especially important to study, because its most powerful effect may be seen in the poorest socioeconomic population with RA. Laudable attempts to study the epidemiology of RA in Britain have been set up. One example is the Norfolk Arthritis Register. However, we would suggest such populations, in which there are a large proportion of higher socioeconomic groups, are unrepresentative of the large industrial cities in Britain. In 239 patients with RA in the Merseyside region under hospital follow up, the social class of our patients was identified using the Office of National Statistics classification of occupations.[7] The patients with RA in Merseyside were of significantly lower social class than the patients with inflammatory polyarthritis studied in Norfolk.[8] Table 1 summarises these findings. If the findings reported by Maiden *et al*[1] are supported by further studies, there would seem to be significant differences in incidence, severity, and mortality in RA according to socioeconomic profiles. This would mean that increased resources should be allocated to regions of greatest need and not, as at present, to areas where socioeconomic class is highest, such as the south of England.

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Table 1

	Social class† 1–2 No (%)	*Social class 3N–3M‡ No (%)*	*Social class 4–5 No (%)*
Inflammatory polyarthritis cases Norfolk and Norwich[8] (154)	51 (33)	73 (47)	30 (19)
RA cases Merseyside (239)	28 (12)*	87 (36)**	124 (52)*

*p<0.00001; **p<0.05.
†Social class based on the Office of National Statistics classification of occupations.[7]
‡N = non-manual; M = manual.